

# Internal Medicine

**Topic :**     **Bronchial Asthma**

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GROUP



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DeSiGn By :

HBX  
هبة خوري

Dr said that you don't have to know any thing except the slides , so it is your only reference for lecture

### Slide 3

Definition:

- A chronic inflammatory disorder of airways.
- Causing recurrent \ intermittent episodes of ( we usually ask about these symptoms ) :
  1. wheezing
  2. breathlessness
  3. chest tightness
  4. cough particularly at night
- Symptoms are worse at night and \or in early morning.

### Slide 4

- Variable airflow limitation “that means the physiology of asthma includes narrowing of airways and this is a variable, in the morning is the worse, afternoon is better, and so on” It goes back to normal either spontaneously or with treatment (reversible.)
- Airways hyper-responsiveness to a variety of stimuli. For example normal people can use clor and there is no problem but asthmatic patient will have a problem

### Slide 5

- Asthma is a common problem affects 14-15 million people.
- More common in females/blacks/inner city.

## Slide 6

- How asthma affects the general population. It is a common disorder, affects 35% (doctor said 30%, he was not specific about numbers, he just emphasized that asthma is a common problem and about 23% of population do not have it, and the rest are affected or past history of asthma) .

## Slide 7

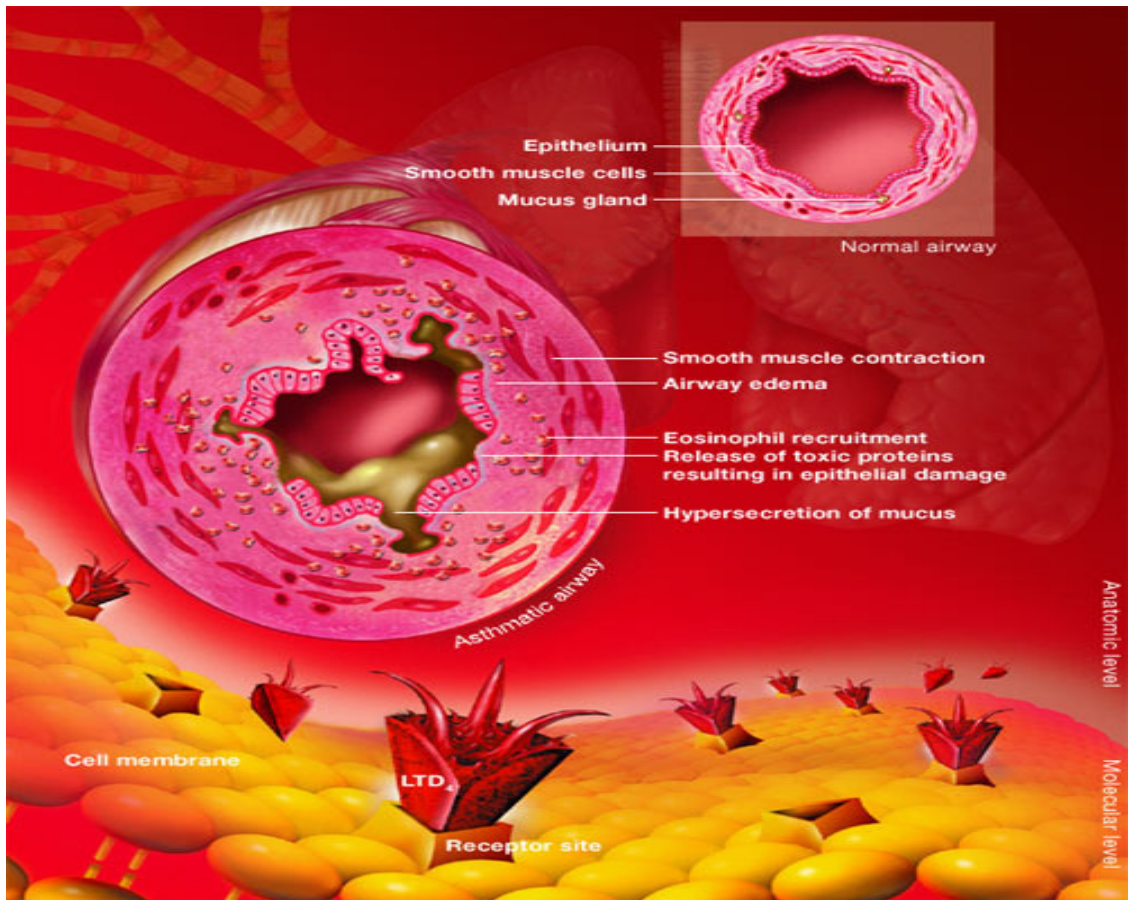
- Asthma is a combination of
  1. genetic factors such as Atopy (حساسيه) . what is the meaning of Atopy ? it means that my system is programmed to be allergic to something .
  2. Environmental factors ( that is why asthma is more common in inner city ) such as Allergen , Viruses , Diet , AB , Smoking .

So it is multifactor process .

## Slide 8

- If you look to this diagram (I don't want to make it complicated مش مطلوب منكواكيد ) but there are several pathways are involved in asthma which make the target of treatment is difficult , so you want something blocks all these ways .

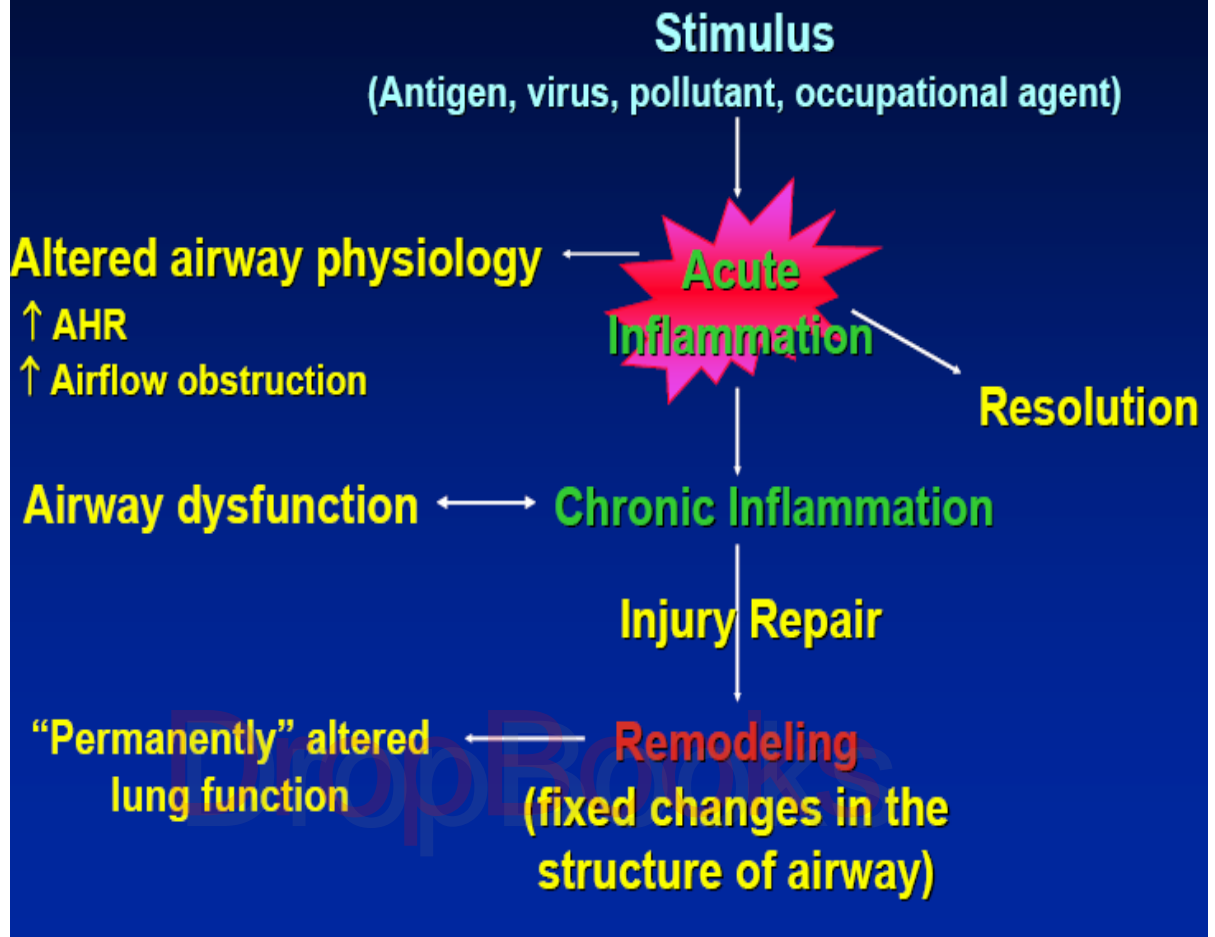
## Slide 9



- If you look to normal airway and asthmatic airway you will find difference between them.
- In asthmatic airway there is thickening, it is not only just mechanical obstruction, if you notice the wall itself is thick.
- Hyper secretion of mucus in lumen. There are glands.

So it is not a symptom like a door open and close No you have to target the inflammation also.

## Consequences of Inflammation in Asthma



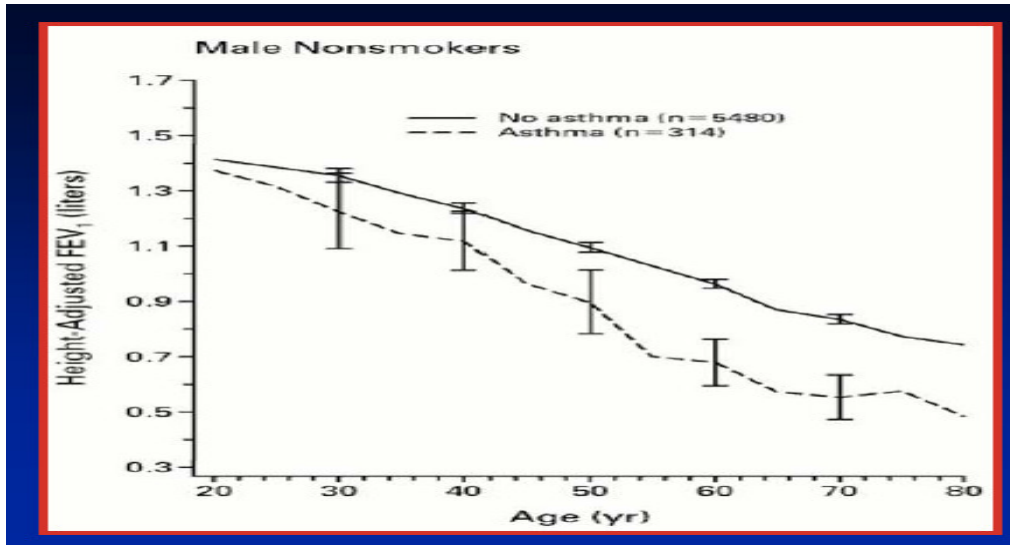
This is **Acute inflammation** that leads to hyper responsiveness and airway obstruction. The resolution may happen if there is treatment for one or two months.

Acute inflammation can progress to **Chronic inflammation** in which the patients suffer from modification (we call it **Airway dysfunction**) and they become asthmatic patients.

Asthmatic patients with no treatment will get **Remodeling** (the airway will experience fixed changes and will not response to treatment that is why we have to treat the patient asthmatic patient before injury).

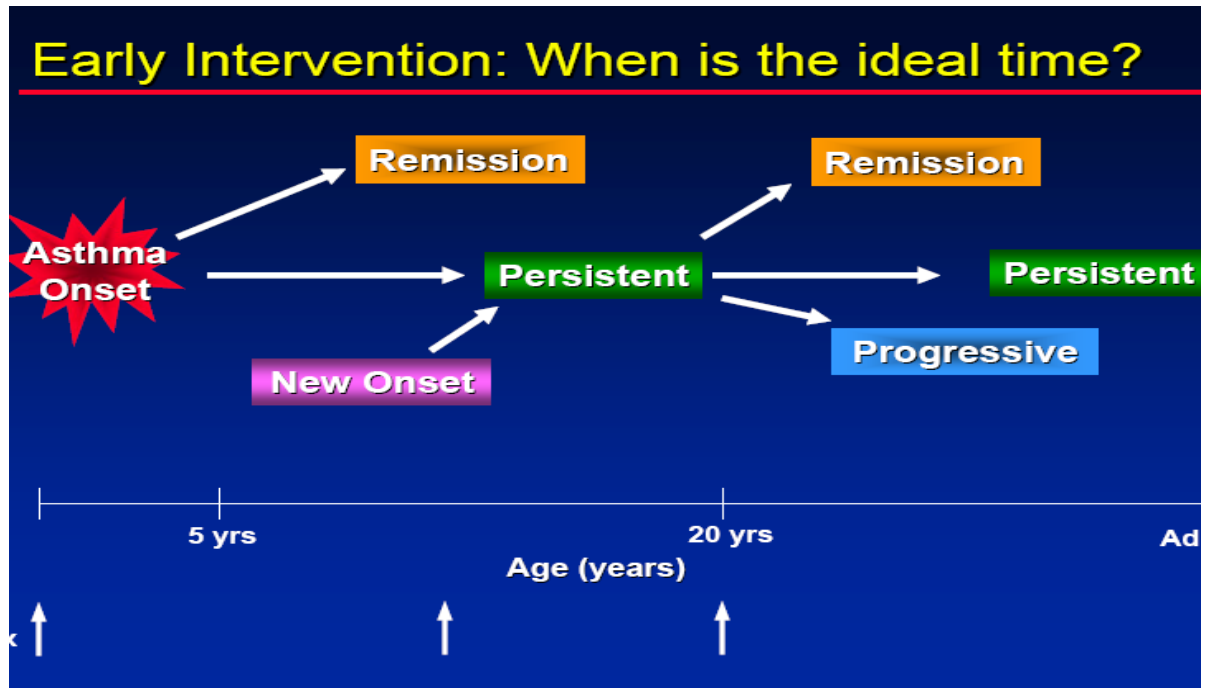


## Slide 11



- This is for nonsmoker.
- FEV1 (Forced Expiratory Volume in 1 second) : maximum amount of air expired in 1 second measured by device . if there is obstruction the patient cant expire all air but if the airway is open he will .
- FEV1 is small that means there is obstruction.
- If you look at normal nonsmoker (no asthma) FEV1 decreases with age, but asthmatic patient their FEV1 is deteriorated which means there is injury, destrtruction, and fixed changes.

Slide 12



It is a common question in clinic what happens after asthma onset?

I'm very careful how to answer this question, but we know if we have early onset asthma a lot of cases will be remised, that is why we say for children's parents the asthma will disappear when they grow up.

Asthma onset at older age → most of patients will have progression and some of them will have remission.

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**Risk Factors for Asthma**

- Allergy/Atopy
- Family history of asthma/allergy
- Perinatal exposure to tobacco smoke
- Early viral respiratory tract infections
- Low birth weight
- Environmental pollution
- Low socio-economic status

- Passive smoking: the inhalation of nonsmokers of the smoke from other people's cigarettes.

Slide 14

### **Atopy and Asthma**

This is also important to know that the most common indoor allergen is House dust mite; it is a mite that feeds on house hold detritus, which often highly allergenic.

Slide 15

### **Indoor Air Triggers**

- Environmental tobacco smoke
- Cockroaches
- House dust mites - common
- Animal dander - cats
- Mold (العفن)

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### **Outdoor Air Triggers**

- Particulate matter (air pollution)
  1. Combustion products
  2. Industrial emissions
  3. Vehicle exhaust
- Outdoor pollens
  1. Olive in JORDAN

Slide 17

### **Additional Triggers**

- **Viral upper respiratory infections**
- Exercise and hyperventilation
- GERD (Gastro-esophageal reflux disease )
- **Sinusitis and rhinitis**



- Diet
- Cold air
- **Drugs**, this is important for you as dentist when you give patient pain killer
  - Aspirin, NSAID, beta blockers

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### **Asthma Diagnosis**

- History and patterns of symptoms (wheeze ,chest tightness, cough ,shortness of breath )
- Physical examination
- Measurements of lung function
- Measurements of allergic status to identify risk factors, there is something called skin test is applied to skin in order to observe the patient's reaction to specific allergy.

DropBooks

Slide 19

### **Symptoms and Signs**

- Variety of symptoms
  - wheeze
  - shortness of breath
  - chest tightness
  - cough
- Asthma symptoms tend to be:
  - Variable and intermittent
  - Worse at night
  - Provoked by triggers

## Slide 20

### Additional Elements in History

- Personal or family history of
  - Asthma
  - Atopic condition: eczema, allergic rhinitis
- Worsening of symptoms after
  - Exposure to recognized triggers
  - Taking aspirin, NSAID, b-blockers
  - Exercise

## Slide 21

### Physical Signs of Asthma

- During exacerbations
  - Wheeze, silent chest (when you put stethoscope on chest you cant hear any sounds due to obstruction ), hyper-resonant
- Wheeze: are diffuse, polyphonic, bilateral and particularly expiratory
- Chronic asthma may have signs of hyperinflation with/without wheeze

## Slide 22

### Differential Diagnoses

There are diseases that look like asthma:

- COPD (Chronic Obstruction Pulmonary Disease), if someone (60 years) is heavy smoker and have symptoms such as wheeze, cough ..., we will think about COPD more than asthma.
- Gastro-esophageal reflux disease (GERD)

- Post nasal drip (allergic rhinitis, sinusitis) :occurs when excessive mucus is produced and accumulates in the back of the nose , it can be caused by rhinitis, sinusitis.
- Cystic fibrosis
- Tumor: Laryngeal, tracheal, lung
- Bronchiectasis
- Foreign body especially in children , if child is wheezed in one side it is more probably to be a foreign body more than asthma
- Vocal cord dysfunction
- Hyperventilation

Slide 23

### **Diagnostic Tools**

#### ■ **Peak flow monitoring by patients**

Peak flow meter is a device used to measure the patient's maximum ability to exhale, peak flow readings are higher when patients are well, and lower when airway is constricted. From changes in recorded values doctor determine lung functionality and severity of asthma symptoms.

#### ■ **Pulmonary function testing (spirometry)** , it will show if there is obstruction.

#### ■ **Bronchoprovocative challenge**, this is for patient claims that he has asthma. we don't have approve; we give him material causes asthma.

Slide 24

### **Pulmonary Function Test**

#### ■ **Obstructive pattern**

- ↓ Forced Vital Capacity (FVC)
- ↓ Forced Expiratory Volume in 1 second (FEV1)
- $FEV1/FVC < 70\%$

#### ■ **Reversible airflow limitation**

- FEV1 increases **by  $\geq 15\%$**  after inhalation of a rapid-acting beta-2-agonist

Slide 25 وادي الموجب

Slide 26

### **Clinical Control of Asthma**

In past the treatment was not good as this time

- No (or minimal) daytime symptoms
- No limitations of activity
- No nocturnal symptoms
- No (or minimal) need for rescue medication
- Normal lung function
- No exacerbations

So our treatment is improved in good way and current medications will achieve improvement (points above) in at least 40% of patient

Slide 27

### **Levels of Asthma Control**

We can put the patients into three groups (controlled, partially controlled, uncontrolled), you will find most of them here “here → refers to partially controlled but I’m not sure “

Slide 28

### **Asthma Management**

I usually tell the patients this statement

“Although there is **no cure for asthma**, **appropriate management most often results in the achievement of control**”

## Slide 29

### Controller Medications

- Inhaled glucocorticosteroids, the most important one

We said that asthma is inflammatory disease so we need anti inflammatory effect of glucocorticosteroids .

It must be inhaled Not systemic to avoid side effect of steroid.

- Leukotriene modifiers ,especially young patient .
- Anti-IgE, very expensive .
- Theophylline
- Systemic glucocorticosteroids

Long-acting inhaled  $\beta$ 2-agonists

## Slide 30

These are the inhaler devices, the same medication (steroid) but different devices .it just a game of manufactures.

## Slide 31

### Reliever Medications

Reliever Medication will cause bronchodilatation immediately after take it

- Rapid-acting inhaled  $\beta$ 2-agonists
  - **Salbutamol**
  - Systemic glucocorticosteroids
  - Anticholinergics
    - Ipratropium
  - Theophylline
- Short-acting oral  $\beta$ 2-agonists

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### **Treatment steps**

Most important medication for asthma is inhaled glucocorticosteroids( the corner stone of the management of asthma ) ,if any treatment doesn't include inhaled glucocorticosteroids it will be mostly incorrect.

You can give inhaled glucocorticosteroids in high, medium, low, and add other medications to treatment plane.

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### **Asthma Exacerbations**

- Episodes of progressive increase in shortness of breath, cough, wheezing, or chest tightness
- Characterized by decreases in expiratory airflow
- Potentially life-threatening and treatment requires close supervision

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### **Primary therapies for exacerbations**

- Repetitive administration of rapid-acting inhaled  $\beta_2$ -agonist
- Systemic glucocorticosteroids instead of inhaled
- Oxygen supplementation

Closely monitor response to treatment with serial measures of lung function

Slide 35 الدكتور حكي شو بدكوا فيها

Not included

Slide 36

The dental patient with asthma .

An update and oral health considerations

Slide 37

## **ORAL HEALTH CHANGES IN PATIENTS WITH ASTHMA**

- **Increased rate of caries development**
- **Reduced salivary flow**
- **Oral mucosal changes**
- **Gingivitis**
- **Orofacial abnormalities. due to obstruction there will be structural changes**
  - Increased upper anterior and total anterior facial height
  - Higher palatal vaults
  - Greater overjets
  - Higher prevalence of posterior crossbites

Slide 38

### **General Oral Health Care Instructions**

- prescribe fluoride supplements for all asthmatic patients , but especially for those taking B2 agoniss
- instruct patients to rinse their mouths after using an inhaler
- reinforced oral hygiene instructions o help minimize gingivitis
- be warw of bossible need to prescribe antifungal agents for patients who chronically use nebulized corticosteroids (like of there is Candida)

Slide 39 + 40

### **Before Treatment**

1. Shedule appointments for late morning or afternoon assess severity of asthmatic condition
2. Consider AB prophylaxis for immunosuppressed patients



3. Consider corticosteroid replacement for adrenally suppressed patients
4. Avoid using dental materials that may elicit an asthmatic attack
5. Use techniques to reduce the patient's stress :
  - Avoid using barbiturates
  - Avoid using nitrous oxide in people with severe asthma
6. Have supplemental oxygen and bronchodilators available in case of acute asthmatic exacerbation

### **During Treatment**

1. Use vasoconstrictors judiciously
2. Avoid using local anesthetics containing sodium metabisulfite
3. use rubber dams judiciously
4. Avoid eliciting a coughing reflex

### **After Treatment**

1. Be aware that some patients may have an adverse reaction to nonsteroidal anti \_ inflammatory drugs
2. Avoid use of erythromycin in patients taking theophylline
3. avoid use a phenobarbitals in patients taking theophylline

إهداء إلى صديقتي من كلية الصيدلية ربي العمري وغيداء الشواهين

The End of Lecture